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"Observations on the Jugular Pulse with  
special reference to Heart-Block."

--- by ---

ANDREW LESLIE DYKES, M.B., Ch. B.

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Great advances have taken place within recent years in our knowledge of the venous pulse in health and disease.

This is mainly due to the invention of ingenious mechanical appliances by means of which minute variations in the venous pulse pressure can be recognised and recorded.

To the genius of James Mackenzie and others who have worked at this subject Modern Medicine owes a great debt.

The information so obtained has much increased our knowledge of the action of the right auricle and right ventricle.

Certain varieties of heart disease especially those in which the action of the heart is irregular or slow are consequently more clearly understood.

During the eighteen months that I have been a resident medical officer at the Sheffield Royal Infirmary I have had abundant opportunities of observing cases of heart disease.

In most of these jugular pulse tracings were taken.

The instrument with which the observations were made was the Mackenzie Ink Polygraph with two recording styles.

By means of this a simultaneous radial and jugular tracing can be obtained.

The subject being a wide one those cases were especially studied in which the radial pulse rate was either lowered or irregular.

In such cases it was always found that the jugular pulse record gave interesting information.

It is proposed in the following pages to give an account of some of these cases together with the graphic records and the deductions made from them.

Each case will be taken separately with a short account of the history, symptoms and physical signs. In those cases in which a post-mortem examination was obtainable, a brief synopsis of this will be given.

Cases of Heart-Block which are sufficiently rare to justify further detail and to which special reference is made in this thesis are discussed at somewhat greater length and the observations made on these cases will not be limited to the jugular pulse alone.



In the interpretation of the tracings an endeavour has been made to follow as closely as possible the teaching of James Mackenzie.

The position most suitable for taking tracings is the following one.

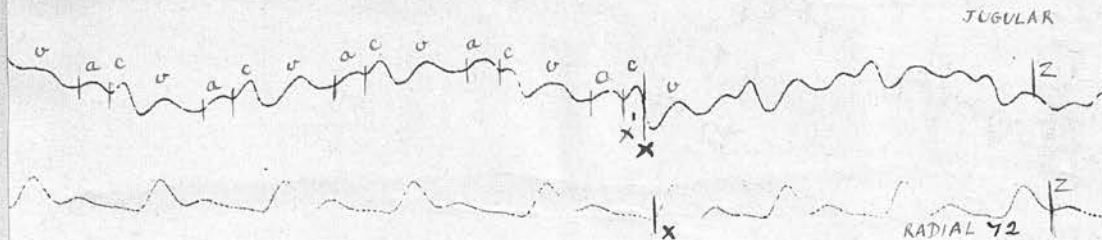
The patient lies on his back with the shoulders slightly raised. The head is supported by a pillow and turned slightly to the right to relax the right sterno-mastoid muscle. All the tracings were taken from the right side of the neck as this gave the best result.

The receiver is placed over the jugular bulb immediately above the inner end of the right clavicle.

Care must be taken to adjust the pressure so that the maximum movement is obtained. Movements of the radial pulse are received by a tambour which is fixed on to the wrist and they are conveyed by means of a rubber tube to a recording tambour.

A brief description of a normal jugular tracing will be given here for the purpose of comparison with those which follow.





A time record is seen at the top of the paper. Each division of this represents one fifth of a second. Beneath this is the jugular tracing and below is that due to the radial pulse.

An abscissa (Z) is taken at the end of each in order to show the relative positions of the recording styles.

From these abscissae points which are simultaneous in each tracing can be obtained by measurement. A perpendicular (X) is drawn at the beginning of a radial pulse beat.

Another perpendicular (X') is drawn on the jugular tracing at an equal distance from the abscissa (Z)

Now it is known that the carotid pulse occurs  $\frac{1}{10}$  seconds in front of the radial.

The perpendicular (X') is drawn  $\frac{1}{10}$  second in front of (X). This is found to fall at the beginning of a small wave which is marked ("C") in the tracing.

Observers are agreed that this wave (C) which is a constant one in jugular tracings is due to

the carotid pulsation.

It will be noticed that this wave has a sharper angle at its apex than the other waves.

A small wave ("A") is seen to precede ("C") in all normal tracings.

This is held to be due to the systole of the auricle.

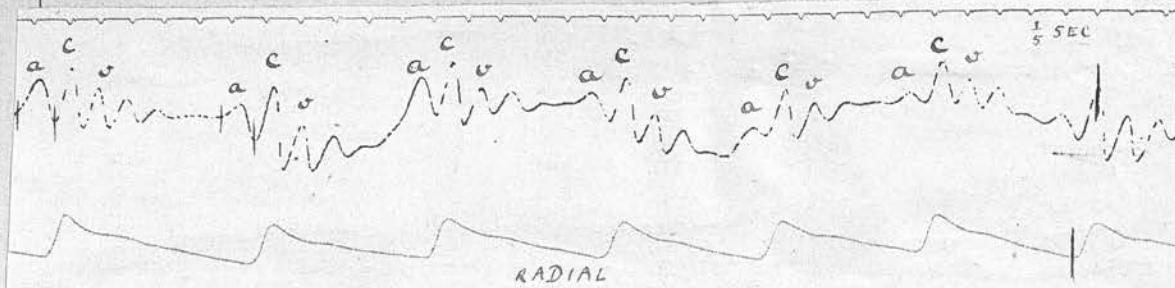
After each ("C") wave another wave marked (V) is seen in the tracing.

This is believed to be due to the storing of blood in the auricle during the time of the ventricular systole. The termination of the rise (V) is due to the opening of the auriculo-ventricular valves. It also occurs at the same time as the dicrotic notch in the radial tracing and hence can be easily identified.

If measurements are taken in the above tracing it will be found that in every case the rise of the wave (V) terminates at the same time as the dicrotic notch.

Sometimes the wave (V) has a notch on it. This is well seen in the following tracing which was taken from a healthy individual.





The cause of this notch has not yet been satisfactorily explained.

It will be noticed in the specimens given that perpendiculars have been drawn at the commencement of consecutive (A) and (C) waves.

The interval between these two lines represents what is known as the A-C interval and is of considerable importance.

The interval is occupied by three events

- (1) The systole of the Auricle.
- (2) The conduction of the stimulus from Auricle to Ventricle.
- (3) The interval during which the ventricular pressure is rising before the opening of the aortic valves.

As (3) is constant any variation in the duration of the A-C interval may be looked upon as a variation of the conductivity of the A.V. bundle.

In the tracings given it will be seen that the A-C interval is either equal to or slightly less than one fifth of a second.



In normal individuals this interval rarely if ever exceeds this time and it is constant in the same individual.

If it does exceed one fifth of a second to any marked extent then one may conclude that the conductivity of the A.V <sup>bundle</sup> ~~node~~ is depressed.

## "STOKES-ADAMS DISEASE."

### Case I.

G. J. a man aged 46 was admitted to the Royal Infirmary Sheffield on November 16th 1908 complaining of attacks of dizziness. The following history was obtained.

A steel-breaker by trade he had been in good health till a fortnight before admission. He then had a sudden syncopal attack one morning while walking up a hill to his work. He did not lose consciousness but simply felt dizzy and was compelled to stand still for two or three minutes after which the attack passed off.

There seems to have been no anginal pain. He tried to do his work that day as usual but felt unequal to it as he appeared to have lost his usual energy. However he persevered for a week and was then obliged to give it up as he found it fatigued him too much and gave rise to a breathlessness which he had never before experienced.

During this week he had no more attacks of dizziness. The following week he stayed at home and while there fainted on three separate occasions. He was told that he was unconscious about  $\frac{1}{2}$  - 1 minute on each occasion. As the feeling of incapacity for work did not leave him and also



on account of the syncopal attacks he applied for admission to the Royal Infirmary.

On inquiry it was found that 9 months previous to this he had had a fainting fit of a similar character which had so alarmed him that he did not go to his work for a week.

When he did so he felt quite well again and from that time to his present illness was apparently in good health.

Patient had never suffered from rheumatism or chorea. Twenty years previously he had had syphilis while in the army and had had a six months course of treatment for it.

Family history revealed nothing. His work was very heavy. It consisted of breaking pieces of steel with a heavy hammer for the purpose.

#### On Examination.

He was found to be a robust well developed man. Beyond that he was rather pale he looked in good health. The only symptoms complained of were the attacks of syncope and the feeling of incapacity for work. He had never had palpitation and only during the fortnight before admission had he noticed shortness of breath on exertion.



The radial pulse rate was 26 per minute. It was irregular in time but regular in expansion.

The rise was rapid, the apex sustained and the fall gradual.

Considerable pressure was required to obliterate the beat. The arterial coats were thickened.



The above is a radial sphygmogram taken on the day of admission.

On examination of the heart the apex beat was felt to be heaving in character. It was situated in the 6th left intercostal space  $4\frac{1}{2}$  inches from the mid-sternal line. A well marked systolic thrill could be felt at the base of the heart.

Deep dulness of heart.

Upper Border at the 3rd rib in the left parasternal line.

### Left Border.

3 inches from mid sternal line in 4th left inter-space.

4 inches       "               "               "       5th       "       "

4½ inches     "               "               "       6th       "       "

### Right Border

1½ inches to right of mid-line at level of 4th rib.

### On Auscultation.

The first sound was replaced by a blowing systolic murmur heard all over the praecordia but most clearly in the aortic region.

It was propagated towards the root of the neck.

The second sound was accentuated in all areas. In the long pauses two or three short flapping sounds could be heard at regular intervals at the base of the heart. They sounded like faint 2nd heart sounds.

The jugular pulsation on inspection was seen to be well marked. Seventy-five pulsations could be counted in the minute.

Unfortunately the polygraph was not available on the day of admission.

There was no oedema of the legs. The other organs on examination appeared healthy.

During the eight days following admission the radial pulse rate varied between 20 and 54 beats per minute. The rhythm varied enormously. As a rule the individual beats were irregular in time.

During one observation the pulse rate was 26 and 5 minutes afterwards it was 36 per minute.

Long pauses frequently occurred.

Sometimes these occurred between every 3, 4 or 5 beats sometimes at longer intervals.



The above tracings illustrate this. The beats between the long pauses were usually quite regular. The patient was noticed to get paler during the pauses and if the pause was very long he lost consciousness for several seconds.

Many attacks of unconsciousness were witnessed and all of them presented similar features.



The face suddenly became pale and the patient fell back in bed. The breathing became stertorous then ceased altogether for three or four seconds. A flush then spread over his face and consciousness returned. The return to consciousness was usually accompanied by sharp twitching movements in the arms and legs. During unconsciousness the eyes were turned upwards and the pupils were dilated and fixed.

Many times after a syncopal attack it was observed that the radial pulse became regular and more rapid.



The above tracing illustrates this point. It shows a regular pulse rate of 44 per minute.

Three minutes before it was taken the pulse rate was less than 30 per minute and the patient had had a short syncopal fit. Why did this increase of pulse-rate occur?

The A-V bundle received a rest during the long pause which accompanied the fit. This had the effect of improving its function of conduct-

ivity which at the time was depressed. Consequently the pulse-rate was increased after the fit. Whenever the pulse-rate fell below 30 per minute the patient was liable to syncopal attacks and on one or two days he had as many as twenty.

Although the radial pulse varied so much in rate the jugular pulsations were never observed to be less than 65 nor more than 80 per minute.

The following is a chart showing the variations of the radial pulse rate. The observations were taken at 8 a.m. in the morning (marked black) and at 6 p.m. in the evening (marked red.)



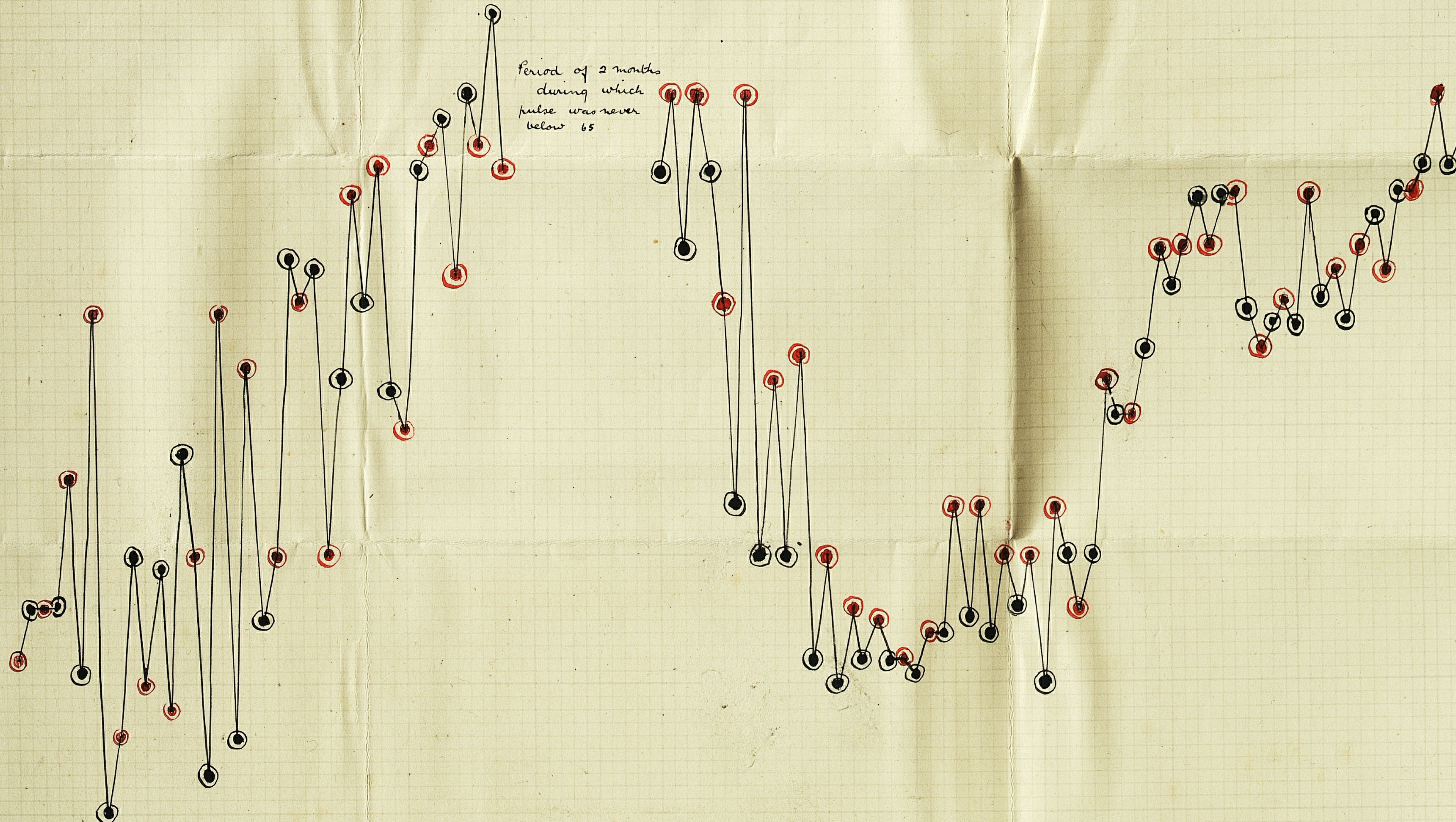
As a rule the radial pulse rate was higher in the evening than in the morning.

It will be observed that 70 per minute was not reached until the patient had been under observation a fortnight. There followed then a period of about 10 weeks during which it was never below 65 nor above 85. The pulse was quite regu-



80  
70  
60  
50  
40  
30  
20

80  
75  
70  
65  
60  
55  
50  
45  
40  
35  
30  
25  
20



DISCHARGED  
2 WEEKS AFTER  
THIS DATE DURING  
WHICH TIME PULSE VARIED 65-75  
BUT NEVER SLOWER THAN 65.

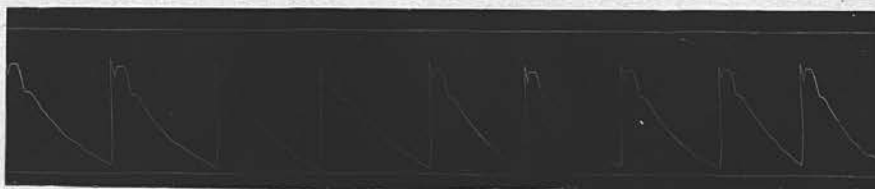


lar during this time and the patient appeared in perfect health.

The pulse rate then again fell by degrees to between 30 and 40 beats per minute and remained so for 12 days.

During this time although the pulse was so slow he only had three very slight syncopal attacks. The rhythm of the pulse was much more regular than was the case during the week following admission.

There were no long pauses noticed which were so common at the first and often the pulse would be for hours quite regular although only 32 per minute.



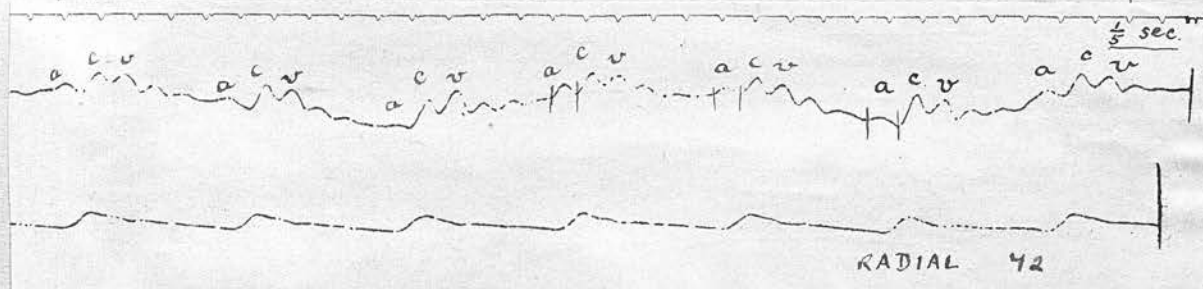
The above sphygmogram illustrates this point. It will be seen from the pulse-chart that the heart gradually regained its normal rate and for a month this was maintained.

At the end of this time the patient was discharged apparently quite well.

After discharge his health was good for a period of 6 months. He even returned to his former work although advised against it. During this time he was seen by me once each week and his radial pulse-rate on these occasions was never less than 70 per minute.

In September 1909 six months after discharge he came up in haste one morning as his former symptoms had returned. He had had a syncopal attack the previous night and he had noticed that his pulse was very slow again. He was readmitted and during the next 12 days his pulse rate varied between 20 and 40 per minute. He had several syncopal attacks just as before and on the 12th day he died suddenly in one of these.

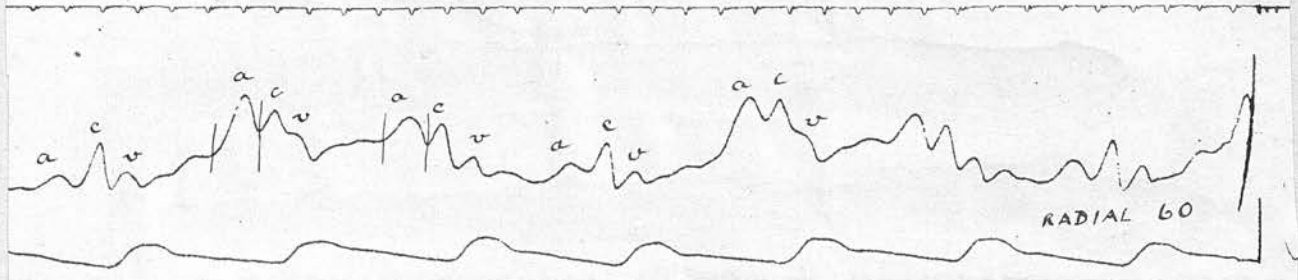
For three days before his death he suffered from dyspnoea even while at rest in bed. Let us now examine the jugular tracings which were taken.



This was taken at a period when the patient was apparently in perfect health. It shows a regular radial pulse of 72 per minute and the jugular tracing is normal in every respect. The

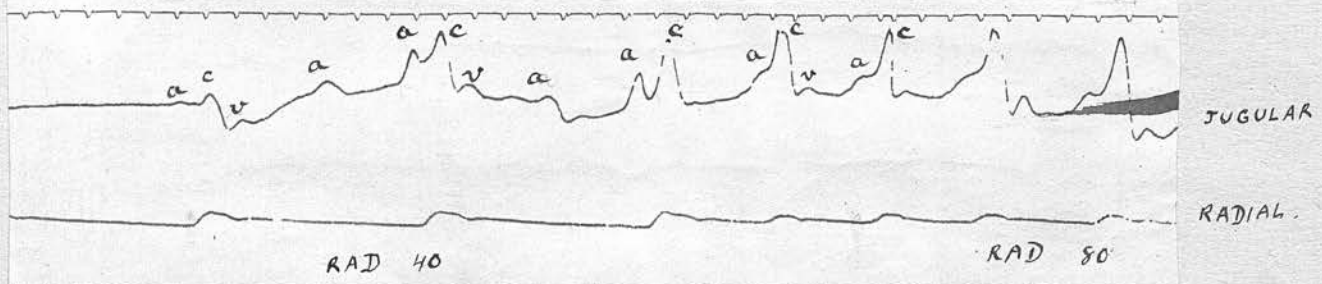


A.C. interval is seen to be about  $\frac{1}{5}$  second. The V wave is double.



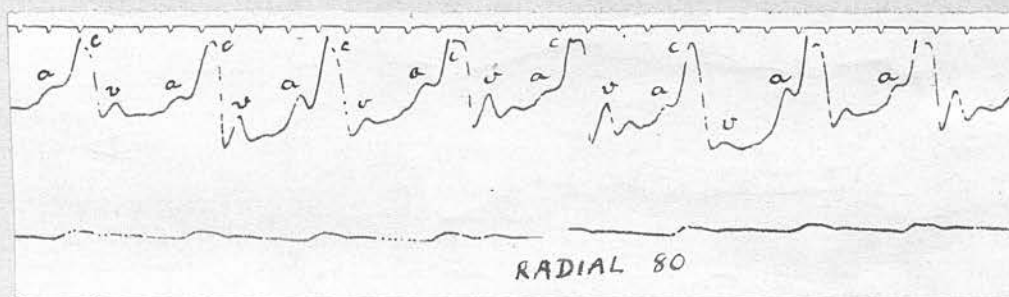
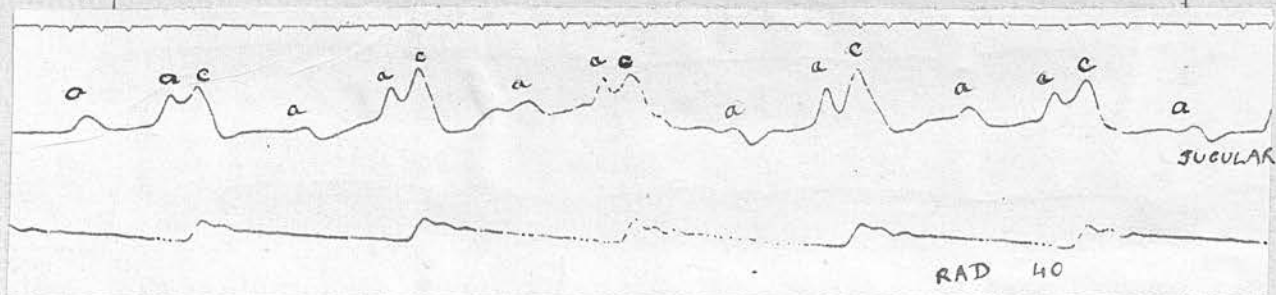
In this we see the radial pulse is 60 per minute and still regular. The jugular tracing shows an A.C. interval greater than  $\frac{1}{5}$  of a second. Hence at the time this tracing was taken there must have been slight depression of conductivity.

The following tracings were taken one day during which the heart showed a very peculiar condition. The radial pulse after beating regularly for two or three minutes at 40 per minute suddenly changed its rate to 80 per minute, after which it again assumed the slower rate. This was observed to occur several times during the day.



This tracing shows the sudden doubling in the pulse rate.





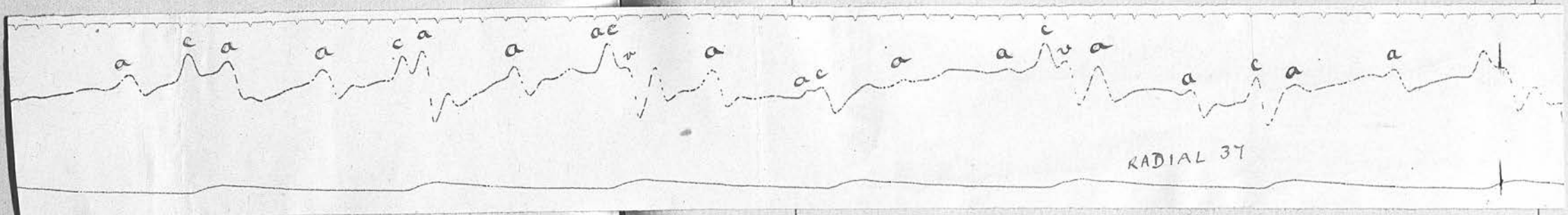
The last tracing was taken  $\frac{1}{2}$  a minute after the one preceeding it.

It will be seen that during the slower rate there are two auricular beats to one ventricular beat. The sudden doubling of the radial pulse rate is caused by the change to the normal condition.

During the slower rate the A.V. bundle is rested and its conductive power restored so that it can again perform its function normally. The sudden change from the slower to the quick rate is interesting as one would expect a gradual restoration of conductivity and hence a gradual quickening of the rate instead of a sudden one as occurred here.

ECG tracing showing a regular rhythm. The waves are labeled 'a' and 'ca'. The tracing is identified as 'RADIAL 40'.

Let us compare this with the following tracing which was taken some days later.

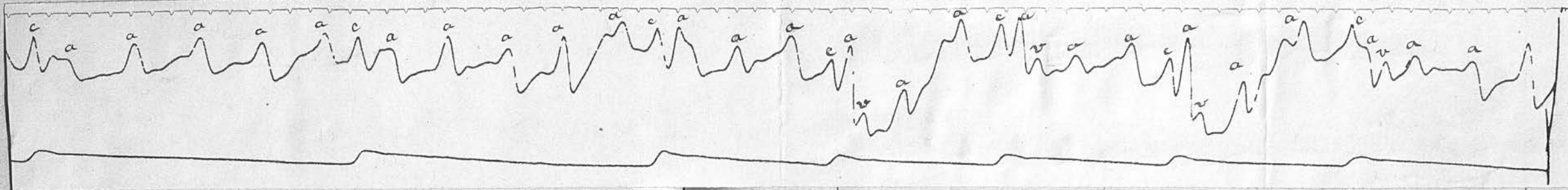


19.

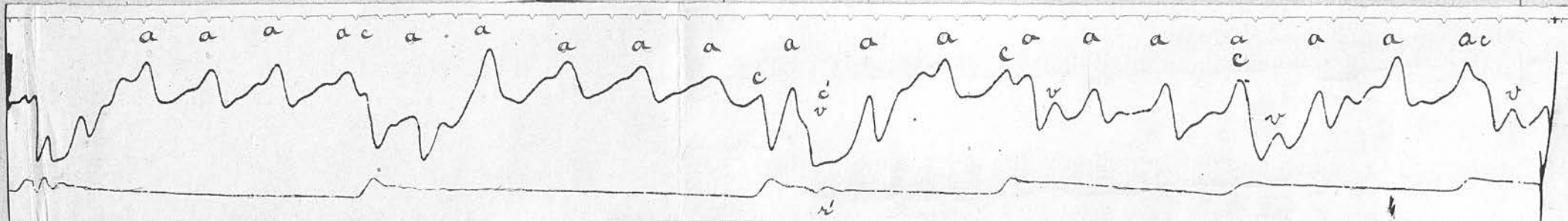


auricles and ventricles are beating independently of each other and the heart-block is complete.

The radial pulse is seen to be about 40 per minute and perfectly regular.



The above tracing shows a ventricular contraction following the fifth auricular one. The ventricular rate is then suddenly changed so that a contraction follows every 3rd auricular one. Hence when this tracing was taken the ventricles were dependent to a slight extent on the auricles for their stimulus to contract

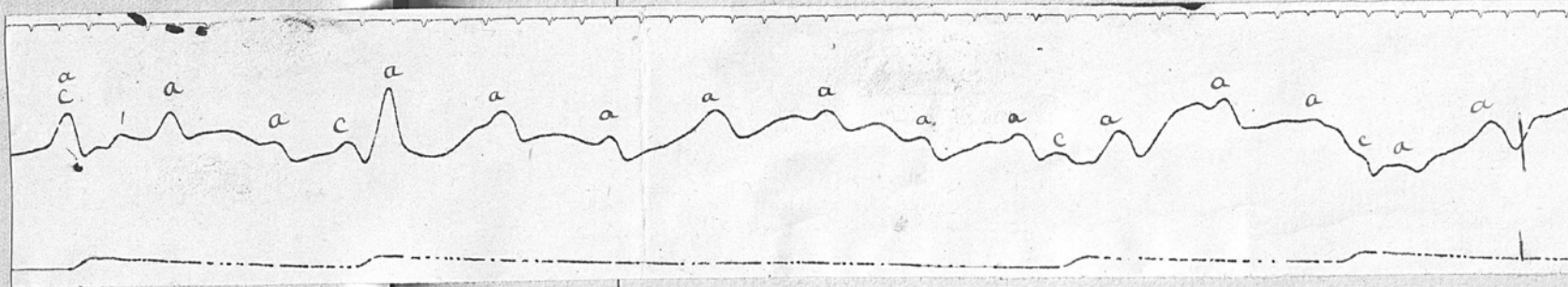


The above is a record of complete heart-block. It is seen that there is no relationship between the "A" and "C" waves and on one occasion they occur at the same time. It also shows an extrasystole of ventricular origin marked R' in the

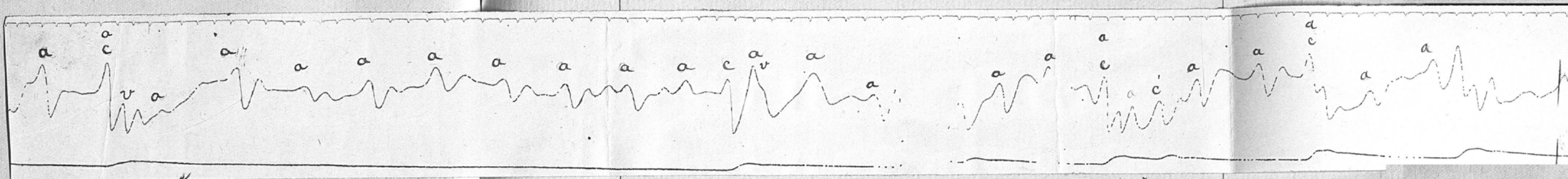


radial tracing.

The occurrence of a (V) wave indicates antecedent contraction of the right ventricle. Hence we may conclude that the ventricles were beating synchronously and at the same rate.



A similar condition is here shown. Seven auricular contractions are seen to occur during one of the long pauses in the radial pulse.



1 beat = 7 sec.

<sup>x</sup> extra-systole.

Nine auricular beats are seen during one long radial pause which was 7 seconds in duration. An extrasystole is seen to occur shortly after. During the day on which this tracing was taken which was 3 days before he died the patient had many syncopal attacks. When the long pause shown on the radial tracing occurred he lost consciousness for two or three seconds but the fit



was a very short one compared to many others which he had.

On several occasions periods of 20-30 seconds were counted during which the patient was unconscious and the radial pulse could not be felt. I should like to draw attention to the frequent occurrence of ventricular extra-systoles in this case. They appeared to be independent of the radial pulse rate as they were noticed when this was normal and also when complete heart-block was established.



Their presence suggests that the portion of tissue in the auriculo-ventricular bundle which gave origin to the independent ventricular contractions was in an irritable state.

It is possible that the degenerative process which had destroyed one part of the bundle and caused complete heart-block rendered another part

more irritable and gave rise to ventricular extra-systoles.

Another interesting feature was presented by this case at a certain stage.

It was noticed during a period of 2 or 3 days preceeding the development of complete heart-block. The pulse had been between 70 and 80 for about ten weeks. It then began to get slower and more irregular in rate before finally falling to 30 beats per minute.

It was during these 2 or 3 days while the radial pulse varied between 50 and 60 beats per minute that the following observation was made. The radial pulse became distinctly slower shortly after the patient coughed.



x  
PATIENT COUGHED



x  
PATIENT COUGHED.

The slowing occurred on the 4th or 5th beat after the coughing. It affected only 3 or 4 beats after which the pulse again became as before. This feature was only present at the period named.



It was absent when the block was complete and when the radial pulse rate was normal.



This tracing was taken when the heart-block was complete. It shows that the rate was unaffected by coughing.

I think the following is the explanation. The coughing gave rise to stimulation of the vagus which acting on an A.V. bundle of which the conductivity was already depressed caused a transient heart-block.

Stimulation of the vagus was ineffective when the conductivity was unimpaired and also when the heart-block was complete.

From this it may be concluded that the vagus cannot exert a direct influence on the ventricles. It can influence them only through the A.V. bundle and is especially liable to do so when the conductivity of this bundle is depressed.

In this thesis notes will be given of two other cases of heart-block in both of which the block was partial.

In these no apparent effect was produced on stimulation of the vagus by means of coughing.

The following is a short report of the post-mortem examination which was made in the case which has just been described.

The Heart.

Pericardium.	Healthy.
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Right Auricle.

Epicardium.	Healthy.
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Orifices of Superior and Inferior Venae Cavae.	Dilated.
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Orifice of Coronary Sinus.	Dilated.
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Fossa ovalis.	Closed.
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Pectinate muscles.	Hypertrophied.
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The auricular wall was hypertrophied and its cavity dilated.

Right Ventricle.

Epicardium.	Healthy.
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Tricuspid orifice.	Admitted 4 fingers.
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The cusps appeared	Healthy.
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Papillary muscles.	Hypertrophied.
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Chordae tendinae.	Thickened.
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Columnae carnae.	Thickened.
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Cavity of the ventricle.	Dilated.
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Septum ventriculorum.	Hypertrophied.
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Cusps of the pulmonary valve.	Healthy
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### Left Auricle.

Pulmonary Veins.	Dilated.
Muscle wall of auricle.	Hypertrophied.
Auricular cavity.	Dilated.
Auricular appendix.	Distended.

### Left Ventricle.

The mitral orifice admitted 3 fingers.

The cusps appeared healthy.

Length of left ventricular cavity along septal wall, 90 m.m.

Length       "       "       "       "       "

lateral wall, 104 m.m.

Ventricular wall.	Hypertrophied.
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"       Cavity.	Dilated.
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At the extreme apex the wall was 6 m.m. thick

At its widest part it was 19 m.m. thick.

Cut sections of the ventricular wall showed the presence of marked interstitial myocarditis (See microscopic slide.)

### Aortic orifice.

The three cusps were much thickened, and contracted.

Their contiguous sides were adherent. On their free margins were several warty formations which are seen in the photograph.

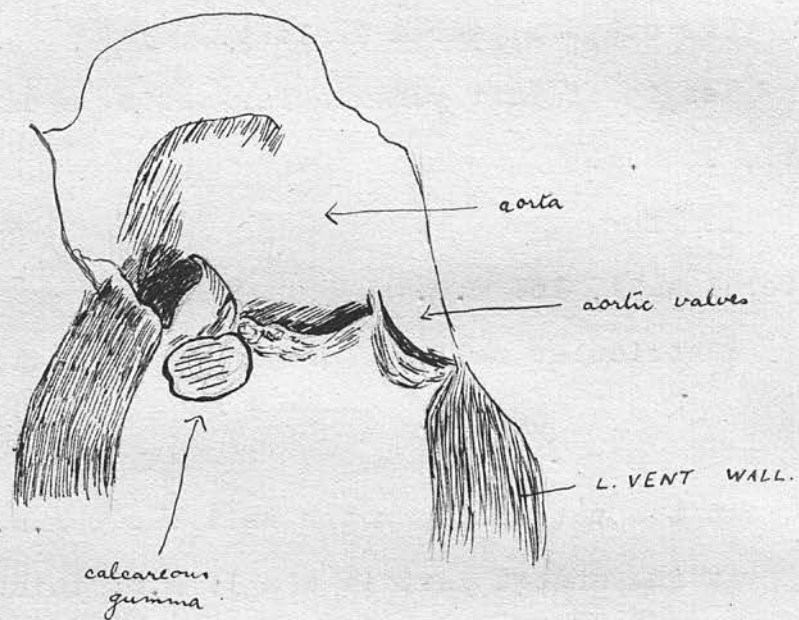
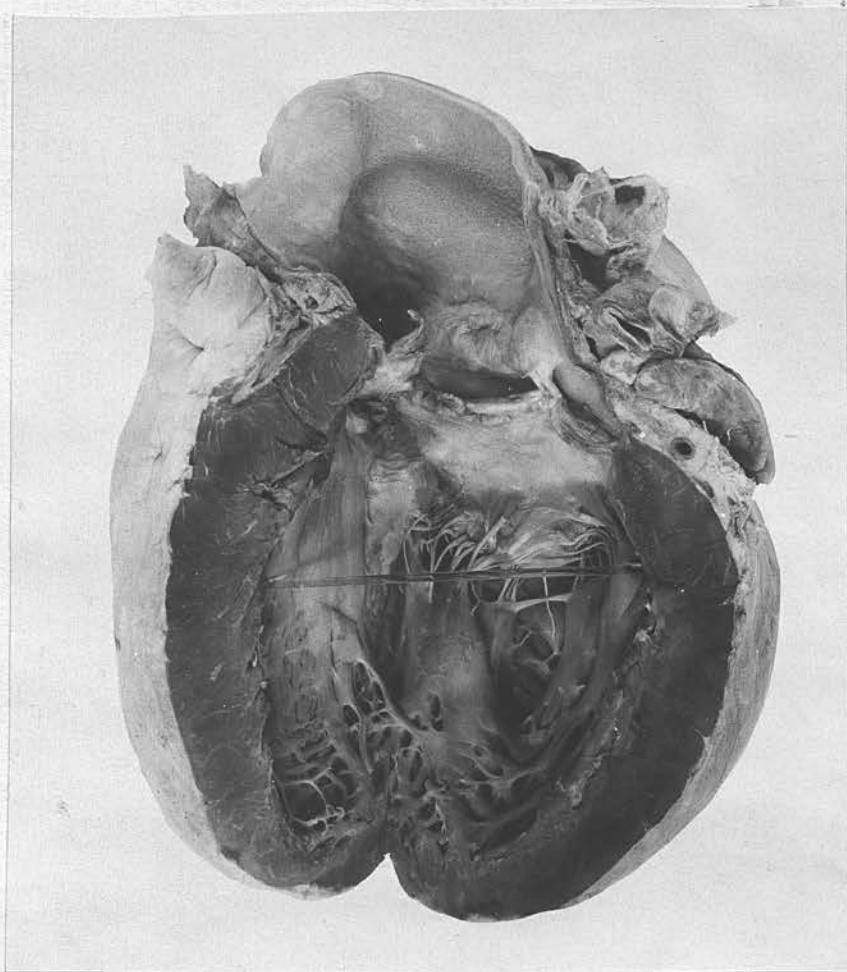


Diagram of photograph to illustrate position  
of gumma.



A thick gritty calcareous formation about the size of a large pea was seen just below the anterior and right posterior cusps. It extended to the lowest part of the ventricular surface of these cusps and also into the interventricular wall.



The photograph shows this formation which I think is a gumma which has undergone calcareous degeneration. It also well shows the interstitial myocarditis, and the hypertrophy of the left ventricular wall.

A block of tissue for microscopic examination was cut out from the adjacent portions of the interauricular and interventricular septa which would include the A.V bundle.

(See Allbutts System of Medicine 1909 Vol. VI, Page 133). Large masses of fibrous tissue encroaching on the heart muscle were seen in these sections.

In places the fibrous tissue had undergone calcification and it was only with great difficulty that a section could be cut.

No trace of the main A-V bundle could be found. The walls of the cardiac blood vessels were much thickened (See Microscopic Slide)

Hence in this case I think we may conclude that the heart-block was due to a gumma which had encroached on the main A-V bundle and eventually obliterated it.

Potassium Iodide (90 grains daily) was regularly administered to this patient but it is impossible to say whether the progress of the disease was in any way retarded by it.



## STOKES-ADAMS DISEASE.

### Case II.

J. V. A furnace-man aged 59 admitted in July 1909 complaining of attacks of dizziness.

### History.

The first symptom appeared 2 years before when he began to suffer from attacks of pain over the praecordia.

It was stabbing in character and shot over to the left axilla but never down the arm. It was always worse after a hard days work. The pain was not constant but came on occasionally and after 2 or 3 minutes duration passed off.

He did not faint during these attacks.

The next symptoms appeared a year before admission. They took the form of shortness of breath on exertion and sudden syncopal attacks. These attacks usually came on while he was at his work. Without warning he would suddenly fall to the ground and for a short period consciousness would be lost.

As far as can be gathered from witnesses there were no convulsions on these occasions.

The syncopal attacks were not associated with

the anginal pain and the only discomfort after an attack was a sensation of cold over the body.

For 6 months he had been quite unable to do his work as a furnace-man on account of these attacks.

#### Previous Health.

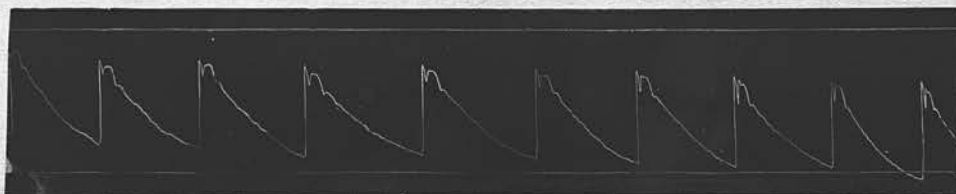
During the 4 years prior to admission he had had 3 attacks of Gout. He had also had an attack of Influenza  $2\frac{1}{2}$  years before. No history of Syphilis could be obtained. He was a man of temperate habits.

#### On Examination.

He was fairly well developed. Height 5 feet 8 inches, Weight 11 stone.

While at rest in bed he was quite comfortable. There was no oedema of legs.

The radial pulse was 32 per minute. It was perfectly regular in force and frequency. The rise was fairly rapid the apex sustained and the fall gradual



It required considerable force to obliterate the beat. The systolic blood pressure was taken several times with a Riva Rocci sphygmometer and



registered 175-190 m.m. of mercury. Sixty-nine pulsations per minute were counted on inspecting the Jugular Pulse.

These pulsations were not quite regular in time.

#### Praecordia.

The apex-beat was felt to be rather heaving in character. It was situated in the 5th left interspace 1 inch external to the nipple line. No thrill could be felt.

#### Deep dulness of Heart.

##### Upper Border.

At level of 3rd rib in the left parasternal line.

##### Left Border.

In 3rd interspace  $3\frac{1}{4}$  inches to left of mid sternal line.

In 4th	"	4	"	"	"	"
" 5th	"	5	"	"	"	"

##### Right Border.

At level of 4th rib  $1\frac{3}{4}$ " to the right of mid sternal line.

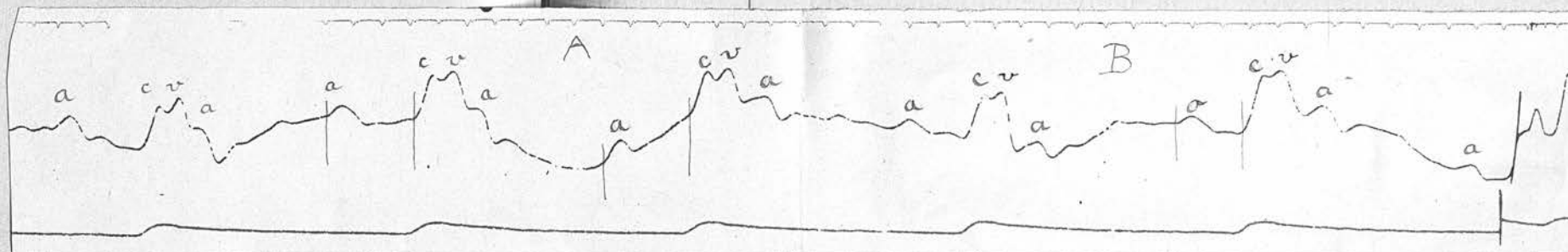
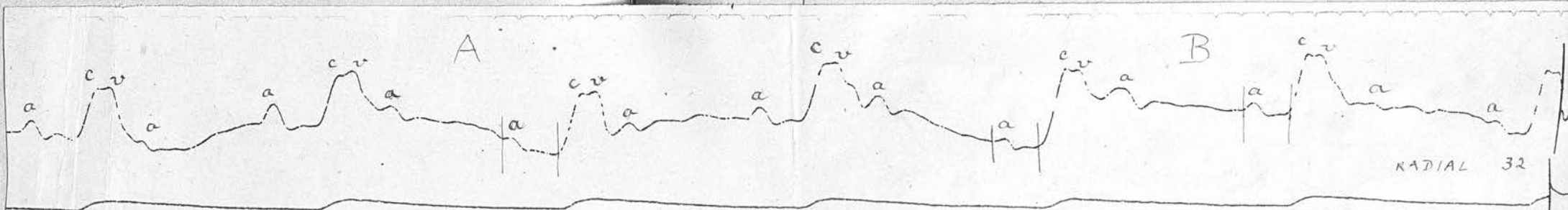
##### On Auscultation.

Both sounds distinctly heard in all areas. No bruits were present.

No signs of disease were detected in the other systems.

During the eight months which have elapsed since the above notes were taken this patient has been under observation.

His condition has rather improved as he has had no syncopal attacks during that time. The radial pulse has never been observed to exceed 40 nor to be less than 30 per minute. Jugular tracings were taken on many occasions and these were all similar. They show a condition of partial heart-block with a 2 : 1 rhythm.



It will be noticed that the waves due to the auricular contractions are twice as numerous as those of the ventricle. Although the C waves occur at perfectly regular intervals the "A" waves



on careful measurement will be seen to occur slightly irregularly.

The interval between the two "A" waves which precede each "C" wave varies slightly in length. At the portion of the tracing marked A this interval is seen to be much less than the corresponding interval at B.

The A.C interval on the other hand at A is seen to be nearly  $\frac{1}{5}$  second longer than at B.

The slight irregularity in the production of the auricular wave is I think due to what Mackenzie terms Sinus Irregularity which is not uncommon in health. When the 2nd "A" wave appears a little earlier the following A.C interval is made longer as the A.V bundle has not had sufficient rest. If however it appears a little later the A.C interval is less as the A.V bundle in this case is more recovered.

Hence it appears that the depression of conductivity has resulted in producing regularity in a radial pulse which would have been irregular had this depression of conductivity not been present.

In this patient stimulation of the vagus by means of coughing or swallowing produced no irregularity in the pulse rate.

## STOKES-ADAMS DISEASE.

### Case III.

T. B. a man aged 70 was admitted to the Sheffield Royal Hospital in October 1909 complaining of attacks of dizziness. He gave the following history. He had been in good health till 4 months before when he had suddenly fainted while out for a walk. His friends told him that he was unconscious for two minutes on this occasion. On recovering consciousness he was able to get up and walk home. He complained of no other symptoms at the time and continued at his work as a shoemaker.

Two days after this he had another one similar to the first.

Since then he had had between twenty and thirty. The loss of consciousness on each occasion was of brief duration. As far as can be gathered there were no epileptiform convulsions. Each attack was preceded by a definite aura. He described it as a tingling sensation which commenced at the top of his head and gradually descended to his eyes at which he lost consciousness.

Between the attacks he felt quite well and was able to do his work. He had not suffered from dyspnoea or pain over the praecordia.



Except that he had had several attacks of Bronchitis his previous health had been good.

He was a married man and his twelve children were all alive and healthy. He had always been temperate in every way.

On Examination.

His appearance suggested good health. He was well developed and well nourished. There was no oedema of legs and while at rest in bed he was quite comfortable.

The radial pulse rate was twenty-eight per minute. It was regular in expansion but not quite regular in time there being one beat in every five or six which had a longer pause than the others.

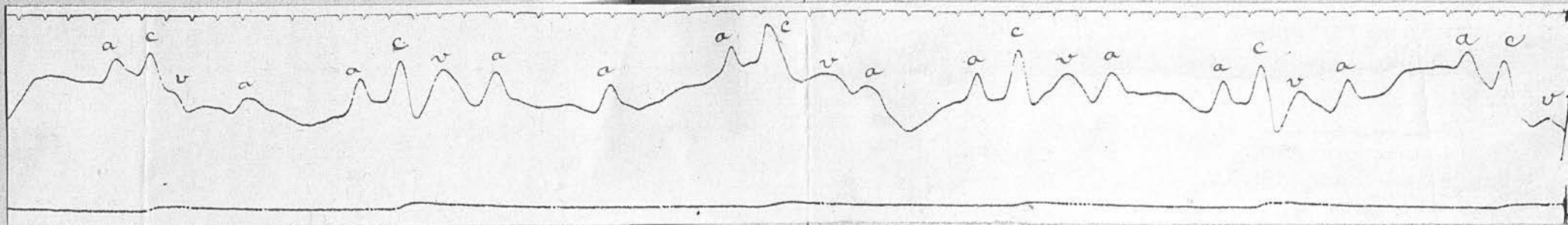
The rise of each beat was fairly abrupt the apex sustained and the fall gradual. The arterial coats were thickened. The systolic blood pressure registered 150 m.m. of mercury, (Riva Rocci.)

On inspection of the jugular pulse between sixty to seventy pulsations per minute were counted. No physical signs of heart disease were detected on examination of the praecordia.

The heart did not appear to be enlarged and the heart-sounds were pure.

Examination of the lungs revealed the presence of emphysema at the anterior margins.

No other signs of disease could be detected.



It will be seen that the auricles are beating regularly at the rate of 60 per minute. One ventricular contraction follows every second auricular contraction except when the longer radial beat occurs in which case three auricular contractions precede the ventricular one.

Hence this is a case of partial heart-block the ventricles being to a certain extent dependent on the auricles for their stimulus to contract.

This patient has been under observation now for a period of six months. His condition during that time has remained the same and he has been able to follow his employment. No syncopal attacks have been observed although he states he has had several on separate occasions when he has been alone. Jugular tracings have not varied.

Beyond the inconvenience of the syncopal attacks he has made no complaint of his condition.



EXTREME BRADYCARDIA Unaccompanied by depression  
of Conductivity.

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Case IV.

T. H. a labourer aged 68 was examined as an Out Patient in August 1909. For 6 months he had been under the care of his own doctor who informed me that on each occasion on which the patient had been examined the radial pulse was observed to be only between 30-35 beats per minute the beats being quite regular in rate and expansion. On enquiry the following history was obtained.

He had been in good health till 6 months previously when he began to suffer from shortness of breath on exertion. This had gradually got worse and for 4 months he had been quite unable to follow his occupation as a labourer. He had never had any syncopal attacks and complained only of the dyspnoea. There had been no cardiac pain and the legs had never been swollen.

Beyond that he had had a severe attack of Rheumatic Fever 25 years previously his health had been good till the onset of his present illness. On examination the patient was found to be well developed and well nourished. While at rest on the couch he had no dyspnoea.

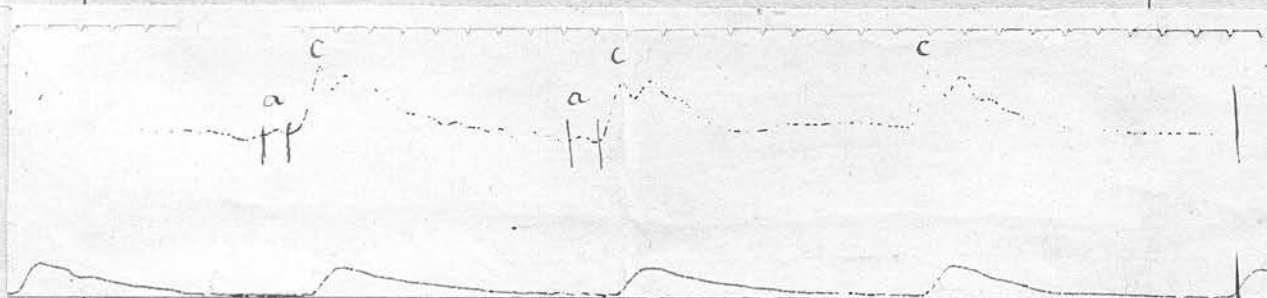
The radial pulse was found to be 32 to the minute. It was perfectly regular both in rate and expansion. The rise was sudden the apex fairly well sustained and the fall gradual. Considerable pressure was required to obliterate the beat. The arterial coats were very thick.

Examination of the praecordia revealed the presence of considerable hypertrophy and dilatation of the left ventricle. A systolic bruit was heard in all areas but was most marked in the mitral area and was propagated towards the axilla.

Systolic indrawing was observed in the epigastric region.

The lungs presented signs of emphysema.

The jugular pulsations did not appear to be more numerous than those of the radial artery. The other organs appeared healthy.



The jugular tracing is not a perfect one but it clearly demonstrates an A.C interval of about  $\frac{1}{5}$  second and one "A" wave only preceeds the C wave.



Through the kindness of Dr. R. Hallam the cardiac movements were observed by means of the X rays and screen. They were seen to be 32 to the minute and quite regular. No part of the heart could be seen to be beating more rapidly than the other.

The patient was again examined 2 months later but no obvious change was noticed in his condition.

He subsequently left the district and no further history could be obtained of him.

I conclude that this is a case of extreme bradycardia unassociated with depression of conductivity in a heart which presented signs of old adherent pericarditis together with mitral regurgitation and its accompaniments.

#### DEPRESSION OF CONDUCTIVITY FOLLOWING PNEUMONIA.

##### Case V.

The patient A.P. a youth aged 23 was admitted to the Royal Infirmary in March 1909 and placed under the care of Dr. Porter. He was suffering from Acute Lobar Pneumonia. The left lower lobe was affected. The patient was well developed and had had no previous illness. The case ran the usual course the crisis being on the 7th day after which he began to improve.

During the febrile stage there were no unusual signs or symptoms. His pulse at that time never exceeded 108 and was regular.

After the crisis the pulse as is usual in such cases slowed down and on the following day was 80 per minute and regular. It continued so for about a week at the end of which time it was noticed that there was some irregularity. There were often long pauses as if a beat had been missed. Sometimes several of these long beats would occur together lowering the pulse rate to 50 per minute. More commonly however a missed beat occurred after a series of four or five regular ones. This state of matters continued till about 4 weeks after the crisis, after which the pulse became again perfectly regular in rate and rhythm and kept so, while under observation during the following month.

The pulse was thus irregular for a period of 3 weeks commencing so a week after the crisis. During this time the slowest rate noticed was 50 per minute and the most rapid was 85.

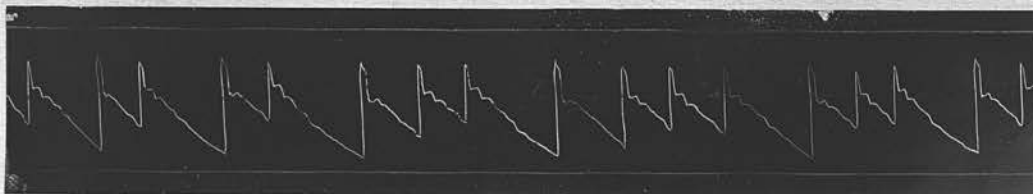
The type of irregularity did not vary much. The pauses never appeared longer than would be accounted for by one missed beat and as a rule one long beat would follow 3, 4, 5, or 6 shorter and regular ones. Occasionally during the three weeks the pulse would be perfectly regular for hours to-



gether but during the day at some time or ~~another~~ the missed beats would appear lowering the pulse rate. The irregularity was usually more marked at night.

On examination the heart presented no abnormal features. There was no enlargement and no bruits were audible.

The following are the graphic records which were taken.



This is an ordinary radial sphygmogram which was taken during a period in which a long beat occurred after every two or three shorter ones. It will be seen that the duration of one of the long beats is about twice that of one of the shorter ones.

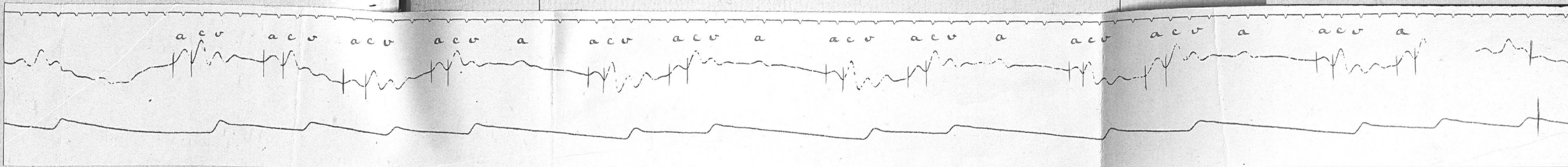
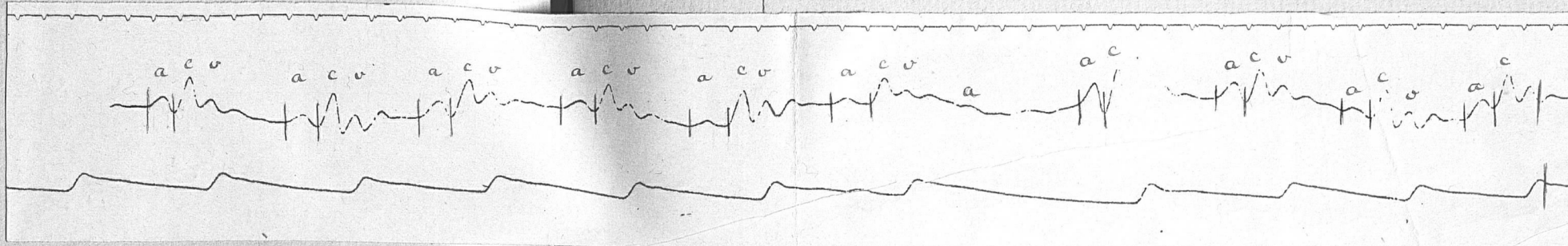
Two explanations present themselves. The long pause may be due to depression of contractility of the left ventricle.

The ventricle receives its stimulus but owing to its contractility being depressed it cannot respond. By the time the next stimulus arrives however it has sufficiently recovered to be able to do so.



The other explanation is that it is due to lowered conductivity.

The ventricle during the long pauses does not receive its stimulus owing to a partial heart-block.



The above tracings show that this is the correct explanation. It is seen that the auricles are beating regularly. During the long pause an auricular wave occurs which is not followed by a "C" wave.

In the first tracing the A.C interval is seen to gradually lengthen until a missed beat occurs.

In the following beat the A.C interval is seen to be about normal ( $\frac{1}{5}$  second).



The missed beat has given the A.V bundle a rest and it has recovered its power of conductivity.

The patient quite recovered and when discharged his pulse had been regular for 3 weeks and a jugular tracing showed an A.C interval of less than  $\frac{1}{5}$  second.

During his illness he did not receive Digitalis which is known to produce a condition similar to this.

The depression of conductivity must have been due to the toxins in some way affecting the A.V bundle, a condition which was evidently quite recovered from.

During the last eight months I have observed many cases of Pneumonia especially so after the crisis was over but I have not found another case showing this condition.

#### "DEPRESSION OF EXCITABILITY OF VENTRICLES

Simulating Stokes-Adams Disease."

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#### Case VI.

G. H. a blade-forging aged 59 was admitted to the Royal Infirmary in January 1910 complaining of Shortness of Breath and attacks of dizziness.

His illness dated from three months before admission when he began to have occasional attacks

Of dizziness brought on by exertion. He had completely lost consciousness several times. On an average two of these attacks occurred each week. They were not attended with anginal pain. Two months before admission shortness of breath on exertion was noticed and his legs became swollen at night. The dyspnoea increased in severity and to this was added a cough which was worse towards evening.

For a month before admission he had been unable to work on account of these symptoms. Beyond that he had had several attacks of Bronchitis he gave no history of previous disease.

#### On Examination.

He was a well-developed man. There was considerable dyspnoea even while at rest in bed and the legs were much swollen.

The radial pulse rate was 40 per minute. It was regular in force and frequency. The expansion was moderate. The rise was abrupt, the apex fairly well sustained and the fall gradual.

Moderate force was required to obliterate the beat.

The arterial coats were thickened and tortuous.

Examination of the heart showed the presence



of mitral regurgitation with considerable hypertrophy and dilatation of both ventricles.

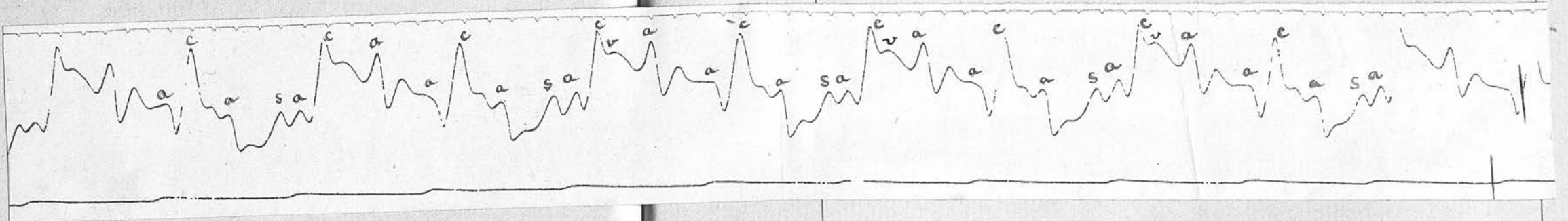
The pulsations in the Jugular Vein were seen to be about twice as frequent as the radial pulse.

The urine averaged 30 ounces per diem and contained a fair amount of albumen.

Examination of the lungs showed that he was suffering also from Chronic Bronchitis.

The dyspnoea gradually increased and he died 2 weeks after admission in a sudden syncopal attack which was the first attack of the kind he had had while under observation. During these 2 weeks his radial pulse varied very little. The rate was usually 40 per minute and the beats were regular in force and frequency.

The jugular pulse tracing was found to be a very constant one.



A marked respiratory wave is seen. Corresponding to every 2nd radial pulse beat a double wave is seen preceeding the "C" wave. This is I

think due to the sinus wave followed by the auricular wave. It will also be seen that an auricular wave appears at regular intervals and that there are two of these to each radial beat.

In other words the auricles are beating twice as frequently as the ventricles.

The A-C interval is seen to be  $\frac{1}{5}$  second.  
Hence there is no delayed conductivity.

This is against the case being Stokes-Adams Disease.

The dropping out of every alternate ventricular contraction must then be due to either

- (1) Depression of Ventricular Excitability.
- (2) Depression of Ventricular Contractility.

No sign of alternating pulse was ever noticed in this case.

This is against (2). Hence I think we may conclude that the missed ventricular beats were due to the failure of ventricular excitability.

The ventricles receive the stimulus regularly from the auricles but owing to the depression of excitability they can only respond to alternate stimuli.

The period of rest given to the ventricles by



the missed beat enables them to recover their excitability so that when the next stimulus does arrive a contraction is produced.

At the post-mortem examination all the cavities of the heart were found to be dilated and their walls hypertrophied. This was most marked in the case of the left ventricle. The mitral orifice was dilated and the cusps thickened and contracted. A microscopic section of the left ventricular wall showed marked interstitial myocarditis. The aorta had several patches of atheroma.

The liver, spleen, and kidneys showed the presence of chronic venous congestion.

Hence I think we may conclude that this was a case in which alternate missed beats were caused by depression of excitability which was due to an interstitial myocarditis to which were added mitral valvular disease and arterio-sclerosis.

It was only by means of a jugular tracing that this case could be distinguished from Stokes-Adams disease.

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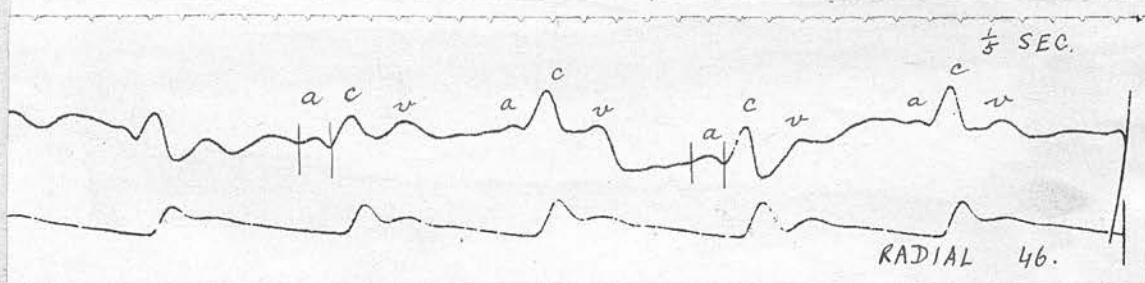
In one of the cases of heart-block described above it was demonstrated that stimulation of the vagus by means of coughing produced slowing of the

ventricular beats.

It is not uncommon to get a slow pulse in certain cerebral conditions such as Compression.

This is believed to be due to stimulation of the vagus.

Several cases of Compression in which the radial pulse was slowed came under my notice. Jugular tracings were taken in each of these in order to find out whether depression of conductivity played any part in the production of the slow pulse. In every case no increase in the A-C interval was observed.



The above was taken from a case of cerebral compression in which the radial pulse rate was 40 per minute.

It will be seen that the A-C interval is normal. Hence no depression of conductivity is present.

A case which I observed in this connection was

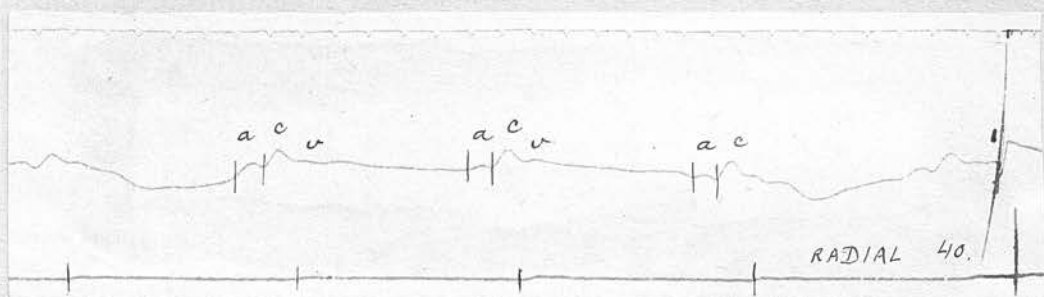


that of a youth aged 20 upon whom the operation of Gastro-enterostomy was performed.

During the 2-3 days preceeding the operation his pulse rate varied between 70-80 beats per minute and was quite regular.

On the evening of the day on which the operation was performed it was noticed that the radial pulse rate although still quite regular was only 40 beats per minute.

It remained so for a week after which the pulse rate gradually increased and reached the normal (70-80) at the end of a fortnight and remained so while under observation.



Jugular pulse tracings showed that the slowing was not due to diminished conductivity.

It may be concluded that in cases where the A-V bundle is not diseased stimulation of the vagus produces slowing of the radial pulse by diminishing the rate of the production of stimuli and not by diminishing conductivity.